with its ligamentous expansion, is essentially the only important structure, from a surgical point of view, on the anterior aspect of the thigh. By this approach it is possible to secure accurate hemostasis of individual vessels with small ligatures, and to avoid mass ligations.

2. The circular incision assures a maximum blood supply to the superficial structures of the stump. The fact that flaps are not fashioned, obviates the necessity for lateral and medial longitudinal incision. Dissection within the remaining soft tissue is avoided, and retraction of these structures en masse, after sectioning, further assures uninterrupted blood supply.

3. The use of a single silk suture to draw the tendons of the detached muscles together over the bone results in an obliteration of dead space. As soon as the effects of the anesthesia have passed, muscular contraction retracts and stabilizes these structures snugly over the end of the bone. The use of only one suture also reduces the amount of foreign material left in the wound.

5. The avoidance of muscle tissue. This has been especially reëmphasized recently by Callander, who has convincingly demonstrated a reduction in postoperative infection and mortality by minimizing trauma to muscle.

6. The end-result is an amputation stump in which the end of the bone is covered not only by skin and subcutaneous fat, but by tendons and deep fascia as well. The anatomical pattern of the remaining thigh is retained, and all soft structures to the end of the stump are movable by voluntary muscular action.

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DISSECTING ANEURYSM*

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ALTHOUGH less than four hundred case reports have been published, dissecting aneurysm is not very rare. Like coronary thrombosis a few years ago, it is now only beginning to be recognized clinically; but the antemortem diagnosis will undoubtedly become common with better understanding of the morbid anatomy and physiology of the condition and the corresponding clinical phenomena.

PATHOLOGY

Dissecting aneurysm is a separation of the coats of an artery by escaped blood. Of the reported cases all but a few were in the aorta, with or without extension to its branches. Three were in the pulmonary artery,[10] and others have been mentioned in cerebral or other smaller arteries. The blood may come exclusively from vasa vasorum;[11] usually the intima is broken secondarily, if not initially. The cleavage plane is in the outer part of the media. If the adventitia is also quickly penetrated at a near-by point, the episode is spoken of simply as *"rupture of the aorta"*: but more or less dissection is the rule in *spontaneous rupture*—not, however, in traumatic rupture. Most dissecting aneurysms soon rupture externally—into the pericardium (more than 50 per cent), left pleural cavity, mediastinum, retroperitoneal tissue, or elsewhere. "Spontaneous" rupture of the undiseased aorta has often been mentioned, with the suggestion that it occurs at "points of lowered resistance," *e. g.*, at the site of a congenital stenosis or at the insertion of the atrophied ductus arteriosus. Two or three centimeters above the aortic valve is the common location. But in the absence of severe trauma, which is rarely alone responsible, it is probable that serial sections would show antecedent disease in most or all cases.

Syphilis is not the cause, and its sclerosing effect on the media may even prevent dissection—witness the patient of Weiss,[12] in whom dissection bordered but failed to invade a syphilitic area. Possibly other infections or obscure atheromatous lesions are at times responsible. But undoubtedly the usual cause is Erdheim's[2] *medionecrosis aortæ idiopathica cystica.* These cysts, possibly of toxic or infectious origin, often multiple, of various sizes but often invisible to the naked eye, are filled with collagenous or necrotic material, and in their formation involve the destruction of medial tissue. They are found at times in the absence of dissecting aneurysm.[9] The tears of the intima which they cause are occasionally multiple,[8] and often appear as slits at a right angle to the direction of blood flow, *i. e.*, favorable to dissection. The aneurysm usually does not embrace the entire circumference of the aorta.

Dissection may progress gradually or intermittently, at times with arrest, thrombosis, organization, and repair; or it may rush through the entire course of the aorta and into one or more large branches. Branches may be choked, or small ones torn off in the process. External ballooning may compress neighboring structures, a remarkable example being that of George II of England, whose dissecting aneurysm compressed the pulmonary artery with resultant rupture of the right ventricle—the catastrophe while straining at stool.[6] And the separated inner coats bulge into the lumen of the vessel and may more or less completely block it, reducing circulation below and raising pressure above. Occasionally this diversion of pressure forces the aneurysmal blood back into normal channels by rerupture into the lower aorta or into one or more of its branches. A few have survived after such an escape-valve effect, the new channel in time becoming endothelialized and carrying part or most of the stream.

DIAGNOSIS

The clinical picture of dissecting aneurysm, with its pain, anxiety, collapse and generally shock-like appearance, has been so commonly confused with that of coronary thrombosis that, in the following description, comparative remarks on the latter condition are included parenthetically. Most of the patients with dissecting aneurysm are middle-aged men, but many are under thirty-five. (Coronary: rare under thirty-five.) Three or four per cent of the reported cases were in advanced pregnancy.[3,4]

* Read before the Section on General Medicine of the California Medical Association at the sixty-ninth annual session, Coronado, May 6-9, 1940.

(Coronary: no such pregnancy incidence.) Hyperpiesis is common. (Coronary: a smaller proportion with hyperpiesis.) Symptoms may begin when the patient is at rest or, more commonly, during exertion, which, however, may not be great. In seven reported cases the onset was associated with defecation, a pregnant woman was seized with pain while stooping, one patient when boarding a bus, another when tolling a bell,[10] Middleton's[5] patient while removing an overcoat, one of Davy and Gates'[1] while stretching the arms backward, one of mine while reaching high up to insert an electric bulb, and another (patient of Dr. K. O. Haldeman) while leaning forward over a table—all suggesting contortion of the thorax as a precipitating factor. (Coronary: onset usually independent of any special exertion.) Early faintness or syncope is common, possibly reflex from the aortic depressor nerve originating at the base of the aorta; and with this may be nausea and vomiting. (Coronary: weakness, slowly developing, is common, but not initial syncope or vomiting.)

The pain may be little, discontinuous, or even absent, but is usually terrific; it may begin mildly and go on with cruel crescendo, but often strikes with explosive violence. Patients may cry out or writhe in agony. They use strong adjectives to indicate the intensity of the pain, but are often at a loss for similes to depict its quality. It may be stabbing, tearing, bursting, etc. Throbbing of the pain in synchrony with the heart beat was admitted by two recent patients. This is easy to understand when successive pulse waves are splitting the media bit by bit, or stretching the newly formed aneurysmal wall; and it may prove to be characteristic. Nitrites may or may not alleviate temporarily. A free interval of hours, days, or longer, often precedes the final rupture. (Coronary: pain may be absent, usually severe with crescendo, often with compression quality, and often preceded by effort-induced angina; attitude usually immobile—patient may sit up or pace the floor, but does not writhe. Pain usually not relieved by nitrites.) The pain commonly begins under the xyphoid and spreads up along the sternum and to the interscapular region; and, with advancing dissection, it is likely to spread down through the abdomen and lower back, perhaps to the legs. As a rule it does not enter an arm unless by cutting down the blood supply. But there are great variations. Radiated pain may open the scene, as in one man who went from his business desk to his exodontist when seized with pain in both lower bicuspids. The dentist promptly sent him to my office in the same building, and by that time the pain was spreading throughout the chest, back, and abdomen. Necropsy the same day showed a tear in the ascending aorta with dissection all the way down and into both iliacs; rupture into the pericardium. (Coronary: pain occasionally epigastric, usually substernal, usually radiating to left arm or both arms, neck, jaw, or interscapular region; may spread, as from lower sternum upward, but not progressively downward as in extensive dissection.)

Depending on the location and degree of bulging into the lumen of the aorta, on involvement of its branches, and on compression of surrounding structures by external expansion or hemorrhage, a great variety of phenomena are displayed. Blood pressure may be low, but is often high and may rise in one or both arms while it falls in the legs. (Coronary: usually a reduced, but well-distributed blood pressure.) Bizarre systolic murmurs or thrills may appear along the course of the aorta or its branches; and about one patient in five suddenly develops signs of aortic valve incompetence from distortion of the cusp anchorages. With slow oozing there may be a pericardial or pleural friction sound, and later signs of fluid—more often in the left pleural cavity, and sometimes serous in character. (Coronary: transitory pericardial friction common, but not fluid accumulation unless with congestive failure.) There may be increase in aortic dullness and tenderness in the suprasternal notch or along the abdominal aorta. And to the many proximal and remote effects of neighborhood pressure characteristic of other aneurysms is here added a great array of possible disturbances from widespread and sudden choking of nutrient arteries to brain, cord, extremities, and viscera. Among those mentioned are: reduction or absence of femoral pulses, inequality or asynchrony of carotid or arm pulses, partial or complete paralysis of one side of the body or of one or more extremities, paresthesias, loss of sphincter control, tympanites, and hemoglobinuria. The neurologic changes may be inconstant.

The x-ray may furnish important evidence, such as a denser shadow inside the enlarged aortic silhouette, an arcuate protrusion with or without pulsation, dissection enlargement of branches, displacement of trachea or esophagus, or indication of escaped blood in a pleural cavity or elsewhere.[18] The usually unmodified electrocardiogram serves to exclude coronary obstruction; but it is possible for dissection at the root of the aorta to close a coronary ostium, and fluid in the pericardium may cause deceptive electrocardiograms. As in coronary occlusion, the acute attack may be followed by fever, leukocytosis, and accelerated sedimentation rate.

PROGNOSIS

About 90 per cent die from rupture in the first few hours or days, and the few survivors are likely to be cardiac cripples because of diminished caliber of the aorta, aortic valve incompetence, arteriovenous communication, or other defect. Osler[7] describes a man who lived comfortably for over thirty years with a double-tube aorta, resulting from dissection and rerupture.

TREATMENT

Liberal use of opiates is in order for pain and restlessness; and blood pressure should be kept constantly as low as possible and tolerable. Erythrol tetranitrate 0.015, if tolerated by the stomach, is desirable for its sustained effect—about four or five hours. Copious venesection seems logical to reduce pressure and blood viscosity and to promote coagulation.

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SOME ASPECTS OF NUTRITION IN SURGICAL PATIENTS

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PART II*

WOUND HEALING

IN a study of wound healing which Smelo[9] made in my department in 1935, he concluded that "factors other than the local dressing appear to play the dominant rôle in determining the rate of wound healing." Anderson,[10] continuing these studies, stated that "the healing of granulating wounds under normal conditions, as determined by precise volume measurement, occurs according to a regular geometric curve which may be expressed in function of area and time by the mathematic equation presented by Carrel[11] and DuNouy[12] for the normal cicatrization of clean surface wounds."

That disruption is still encountered in wounds free from infection in which hemostasis was excellent, in which trauma to tissues and tension were minimal, and unusual strain obviated, strongly supports the concept that other factors of a general character play an important part in the failure of certain wounds to heal. That purely local factors may intensify the factors of a biologic character will not be doubted by anyone who has carried on investigations in this field.

We have shown that dogs which have been made hypoproteinemic by prolonged feeding of a low protein diet and plasmaphereses have a marked delay in fibroblastic proliferation, and thus wound healing is retarded. The hypoproteinemia in our animals was but one manifestation of the protein starvation of the dogs. Although at first we were inclined to attribute the delay in fibroblastic proliferation to the presence of edema, we are now convinced that the mechanism is associated with a profound disturbance in protein metabolism, the hypoproteinemia being only an easily measurable indicator of the extent to which so-called labile stores of protein have already suffered.

It is well known that cellular repair and regeneration require protein, for in the absence of an adequate amount of certain essential amino-acids growth cannot take place. Admont Clark[14] has shown that on a diet high in protein there was no quiescent period in the repair of wounds, and Harvey and Howes[15] have reported that such a diet causes accelerated fibroblastic proliferation. Without adequate building stones, repair cannot take place.

A protein deficiency is, of course, not the only mechanism resulting in wound disruption. Sokolov[16] and Lanman and Ingalls[17] have shown that a vitamin C deficiency is also an important biological factor in this complication. These two nutritional disturbances are frequently found in patients who come for operations for gastric ulcer and cancer, duodenal ulcer, and biliary tract disease. That plasma may be used to replenish depleted protein stores was indicated in the experiments in which we gave large amounts of plasma, as much as 2,400 cubic centimeters, during a two-week period to hypoproteinemic dogs that had been on a low protein diet for some days. The amount of plasma which we administered intravenously was more than six times the calculated plasma volume of the animal, but the plasma protein concentration never exceeded the original normal level for the dog. With the restoration of a normal serum protein concentration and very likely a more nearly normal store of tissue protein, the wounds promptly healed. Addis and his associates[4] and Holman, Mahoney, and Whipple[13] have found that plasma protein can be utilized to replenish the depleted stores of tissue protein, and it is this purpose, we believe, that the excess protein fulfilled.

When all the local factors favoring wound disruption are controlled, there will remain wounds whose failure to heal must be due to more widely acting causes such as hypoproteinemia and a reduction in the labile protein stores, and deficiencies in important accessory foodstuffs.

DIET AND SUSCEPTIBILITY OF THE LIVER TO INJURY

Data which Goldschmidt, Vars and I[18] have collected during the last few years strengthen the impression that the susceptibility of certain tissues to injury by a variety of chemical agents may be influenced by the composition of the organ at the time of injury. We have paid particular attention to this relationship in a single organ—the liver, but the data we have simultaneously collected indicate that the relationship may have a much wider application. That a diet high in carbohydrate is protective, and that a diet high in fat induces maximal susceptibility of the hepatic cells, when the liver is exposed to chloroform, has been re-

* Part I appeared in the July issue on page 10.